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PRURITUS IN VULVAR LICHEN SCLEROSUS: NEUROIMMUNE MECHANISMS AND THERAPEUTIC IMPLICATIONS

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ABSTRACT

Vulvar lichen sclerosus is a chronic inflammatory and fibrosing dermatosis of the anogenital region, in which pruritus is one of the most frequent, burdensome, and clinically relevant symptoms. Although itch in vulvar lichen sclerosus is often interpreted as a direct consequence of local inflammation and epithelial fragility, emerging evidence suggests a more complex neuroimmune phenotype involving barrier disruption, keratinocyte-derived inflammatory signaling, immune-cell infiltration, altered innervation, perineural inflammation, neurogenic inflammation, and possible peripheral or central sensitization. Pruritus is clinically important not only as a symptom of active disease, but also as a driver of scratching, fissuring, sleep disruption, sexual avoidance, anxiety, and impaired quality of life. Topical potent or ultrapotent corticosteroids remain the cornerstone of treatment and are effective for many patients; however, persistent pruritus, pain, burning, or vulvodynia may continue despite adequate control of visible inflammation. In such cases, clinicians should reassess diagnosis, adherence, irritant exposure, infection, genitourinary syndrome of menopause, contact dermatitis, architectural change, and neuropathic pain. Recent studies highlighting perineural inflammation, altered nerve-fiber distribution, quality-of-life impairment, steroid hesitancy, and preliminary responses to neuromodulatory or biologic therapies support a more multidimensional approach to symptom control. This review summarizes current evidence on pruritus in vulvar lichen sclerosus, focusing on neuroimmune mechanisms, patient-reported burden, clinical assessment, and therapeutic implications.

KEYWORDS

Vulvar Lichen Sclerosus; Pruritus; Vulvodynia; Neuroimmune Inflammation; Perineural Inflammation; Topical Corticosteroids; Dupilumab; Amitriptyline

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1. Introduction

Vulvar lichen sclerosus (VLS) is a chronic inflammatory dermatosis that predominantly affects the anogenital skin and may lead to pallor, atrophy, hyperkeratosis, fissuring, ecchymoses, scarring, introital narrowing, clitoral burying, labial resorption, dyspareunia, and increased risk of vulvar squamous cell carcinoma in a subset of patients. Although its pathogenesis remains incompletely understood, current evidence implicates immune dysregulation, genetic predisposition, epithelial barrier dysfunction, chronic inflammation, oxidative stress, hormonal factors, fibroblast activation, and extracellular matrix remodeling. (De Luca et al., 2023; Paganelli et al., 2025)

Among the symptoms of VLS, pruritus occupies a central position. It is often the symptom that prompts medical consultation, contributes to diagnostic delay when mistaken for candidiasis or nonspecific irritation, and strongly affects quality of life. Itch may coexist with burning, soreness, fissuring, pain, dysuria, dyspareunia, and altered genital self-image. Importantly, pruritus is not merely a marker of inflammation; it may participate in an itch–scratch cycle that aggravates epithelial injury, promotes fissuring, increases secondary irritation, and reinforces neuroimmune sensitization. (De Luca et al., 2023; Raef & Elmariah, 2021)

The clinical importance of pruritus has recently been reinforced by outcome-measure initiatives. In the CORALS symptom-domain initiative for vulvar lichen sclerosus, expert consensus identified itch among the key symptoms that should be captured in future research, alongside pain with vaginal penetration, cuts or tears, pain, and irritation. This aligns with patient priorities and emphasizes that symptom control should not be considered secondary to objective lesion improvement alone. (Foster et al., 2026)

Despite its importance, the mechanisms of itch in VLS remain underdefined. In contrast to atopic dermatitis or prurigo nodularis, where specific pruritogenic cytokines and neural pathways have been extensively studied, VLS-specific itch biology is still emerging. Available data point toward a mixed inflammatory, barrier-related, neurogenic, and possibly neuropathic process rather than a single pathway. This review summarizes current evidence on pruritus in VLS, with particular emphasis on neuroimmune mechanisms, quality-of-life implications, and therapeutic decision-making.

2. Methods

This article is a targeted narrative review based on a predefined corpus of publications concerning vulvar lichen sclerosus, pruritus, neuroimmune inflammation, innervation, perineural inflammation, cytokine signaling, treatment guidelines, quality of life, sexuality, steroid hesitancy, and emerging therapies. Studies directly addressing pruritus, pain, vulvodynia, quality of life, sexual dysfunction, treatment adherence, and patient-reported outcomes in VLS were prioritized. Mechanistic studies were included when they addressed neural, immune, epithelial, or inflammatory pathways plausibly relevant to itch. Because the included literature is heterogeneous and includes guidelines, narrative reviews, cross-sectional studies, systematic reviews, qualitative studies, case series, and case reports, no formal meta-analysis was performed. Findings are presented qualitatively, with attention to the distinction between established therapeutic evidence and hypothesis-generating observations.

3. Clinical Phenotype of Pruritus in Vulvar Lichen Sclerosus

Pruritus in VLS may be intermittent or persistent, mild or severe, localized or diffuse, and may involve the vulva, perineum, and perianal skin. It frequently coexists with burning, soreness, fissuring, dyspareunia, irritation, and pain. Some patients describe nocturnal worsening, sleep disruption, inability to avoid scratching, and post-scratch burning or fissuring. Others report a transition from itch to pain or persistent sensitivity even after visible inflammation has improved, suggesting overlap with neuropathic or vulvodynia-like symptoms.

In a histologic case series of lichen sclerosus, most patients with documented symptoms were symptomatic at biopsy; the most common symptoms included pruritus and pain, reported in 57% and 40% of

documented cases, respectively. (Del Papa et al., 2024) These data are consistent with clinical experience in which itch and pain often coexist rather than represent mutually exclusive symptoms.

The symptom complex of VLS is clinically relevant because persistent itch may be both a sign of ongoing inflammation and a manifestation of secondary processes such as irritant dermatitis, candidiasis, contact allergy, urinary exposure, atrophic vulvovaginal changes, lichen simplex chronicus, or neuropathic pain. Therefore, persistent pruritus should not automatically be interpreted as corticosteroid failure. Instead, it should trigger structured reassessment of disease activity, treatment technique, adherence, comorbid vulvar conditions, and neural pain features.

4. Burden of Pruritus: Quality of Life, Sexuality and Psychodermatology

The burden of VLS extends beyond visible skin changes. Pruritus, burning, pain, dyspareunia, fissuring, and fear of worsening may interfere with sexuality, work productivity, daily activity, clothing choices, physical activity, self-image, sleep, and psychological well-being.

A cross-sectional study of 158 women with lichen sclerosus documented substantial impairment in sexual function and quality of life. The mean Female Sexual Function Index score was 13.83, indicating markedly reduced sexual functioning, while the mean Dermatology Life Quality Index score was 7.88, corresponding to a moderate impact on everyday life. The highest DLQI subscores included sexual difficulties and symptoms such as itching, soreness, and pain. The mean WHO-5 score suggested that approximately 40% of participants had signs of depression. (Vittrup et al., 2022)

A systematic review and meta-analysis focusing on sexual dysfunction in lichen sclerosus included 23 studies and reported a pooled prevalence of sexual dysfunction of 59%, with dyspareunia or generalized pain with intercourse being the most frequently reported type of dysfunction. (Pope et al., 2022) Although sexual dysfunction cannot be attributed solely to pruritus, chronic itch contributes to avoidance, anticipation of pain, embarrassment, genital self-consciousness, and fear of fissuring.

A retrospective case-control study of 51 women with female genital lichen sclerosus and 45 healthy controls demonstrated higher depression scores, reduced sexual quality of life, and diminished work productivity among affected women. The study reported that 53% of women with genital LS had a PHQ-9 score ≥10 compared with 16% of controls, and total work productivity loss was estimated at 36.67% among professionally active participants. (Jabłonowska et al., 2023)

Vulvar-disease-specific instruments further emphasize the burden of VLS. In a study comparing vulvar lichen sclerosus, vulvar lichen planus, and chronic vulvovaginal candidiasis using the Vulval Quality of Life Index, patients with VLS had a baseline median VQLI score of 14.00, which improved to 5.00 after individualized treatment. The highest-scoring baseline domain in VLS was future health concerns, indicating that fear of disease progression, cancer risk, and uncertainty may persist alongside physical symptoms. (Wu et al., 2022)

A separate study specifically examined why some long-term topical corticosteroid-treated patients with VLS continue to experience poor quality of life. Poor quality of life was markedly less common in long-term treated patients than in pretreatment patients; however, among treated patients who still reported poor quality of life, the highest scoring domains were sexuality, anxiety, symptoms, and activities of daily living. Poor quality of life was associated with partial treatment adherence, suboptimal disease control, scarring progression, and urinary incontinence. (Wijaya et al., 2022)

These findings support a practical conclusion: successful management of VLS-related pruritus requires more than suppression of visible inflammation. It also requires attention to sexual function, anxiety, adherence, patient education, comorbid vulvar pain, and the psychosocial meaning of chronic genital symptoms.

5. Neuroimmune Mechanisms of Pruritus in VLS

5.1. Barrier disruption and the itch-scratch cycle

The vulvar epithelium in VLS is structurally vulnerable. Atrophy, fissuring, hyperkeratosis, erosions, architectural change, and local inflammation may lower the threshold for itch and pain. Barrier disruption facilitates exposure to urine, sweat, friction, hygiene products, topical medications, and microbial antigens. Repeated scratching further damages the epithelial surface, leading to fissures, bleeding, burning, and secondary inflammation. This creates a self-perpetuating itch-scratch cycle.

In this model, itch is not simply a symptom of inflammation; it is a mechanical and neuroimmune amplifier of disease activity. Scratching can worsen epithelial injury, expose basement membrane zone antigens, promote inflammatory recruitment, and increase peripheral nerve activation. The “outside-in” model of VLS pathogenesis similarly emphasizes that local epithelial damage and barrier disruption may precede or amplify immune activation. (Gupta, 2025)

5.2. Keratinocyte-immune signaling

Keratinocytes are not passive structural cells. They release cytokines, chemokines, antimicrobial peptides, alarmins, and growth factors that regulate local inflammation and may affect sensory nerve endings. Emerging spatial and single-cell transcriptomic studies suggest that keratinocytes are key players in VLS pathogenesis, supporting a model in which epithelial cells participate actively in immune recruitment and tissue remodeling. (Sun et al., 2024, 2026)

The cytokine profile of LS has been described as involving immune activation with a prominent Th1-oriented pattern and inflammatory mediators such as interferon-γ, interleukin-1, interleukin-6, and tumor necrosis factor-related pathways. Cytokine networks may also intersect with extracellular matrix remodeling and fibrosis, creating a link between inflammation, sclerosis, and symptom persistence. (Paganelli et al., 2025)

Although specific pruritogenic cytokines in VLS remain insufficiently characterized, chronic inflammatory cytokine exposure may lower sensory nerve thresholds and enhance neuroimmune crosstalk. This provides a plausible explanation for patients who continue to experience itch or burning despite partial clinical improvement.

5.3. Altered innervation and neurogenic inflammation

Direct evidence linking VLS with altered innervation is limited but important. One immunohistochemical study comparing genital and extragenital lichen sclerosus found differences in nerve-fiber distribution, vanilloid receptor expression, and immune-cell infiltration. The study reported fewer intraepidermal nerve fibers in LS samples compared with healthy skin, but a greater amount of total subepidermal nerve fibers and CGRP-positive fibers in genital LS compared with extragenital LS and healthy controls. Macrophage and mast-cell densities were increased in LS samples, and genital LS had higher macrophage density than extragenital LS. (Vuković et al., 2022)

These findings are clinically meaningful. CGRP-positive fibers are relevant to neurogenic inflammation, vasodilation, pain, and itch. Mast cells can interact with sensory nerves and release mediators that amplify pruritus. Macrophages and T cells may contribute to inflammatory remodeling and cytokine release. Therefore, altered innervation and immune-cell infiltration provide a plausible biological substrate for the intense pruritus and sensory symptoms often observed in genital LS.

5.4. Perineural inflammation

Perineural inflammation represents another possible link between inflammation and sensory symptoms. A retrospective histologic study of 53 LS cases identified perineural inflammatory infiltrates in 15% of cases. The infiltrate was more frequent in male patients, and cases with perineural inflammation showed a statistically significant increase in dermal plasma cells compared with cases without this feature. The study also reported that pruritus and pain were common among symptomatic patients. (Del Papa et al., 2024)

The finding of perineural inflammation should be interpreted cautiously. It does not prove that perineural inflammation causes pruritus, nor does it establish a therapeutic target. However, it supports the concept that nerves may be anatomically involved in the inflammatory process of LS. Future studies should assess whether perineural inflammation correlates with itch severity, pain, treatment resistance, disease duration, sex, anatomical site, and response to neuromodulatory therapy.

5.5. Neuropathic itch and vulvodynia overlap

In some patients, symptoms persist after inflammation is clinically controlled. These patients may describe burning, allodynia, hypersensitivity, spontaneous pain, or vulvodynia rather than classic itch alone. Such symptoms may reflect peripheral sensitization, central sensitization, pelvic floor dysfunction, or secondary neuropathic pain. The distinction matters because escalation of topical corticosteroids may not relieve neuropathic pain and may instead increase frustration, treatment burden, or adverse effects.

A case series of 20 postmenopausal women with biopsy-proven VLS and vulvodynia refractory to standard treatments evaluated oral amitriptyline as an adjunctive therapy. Seventeen of 20 patients reported significant pain reduction, 12 achieved complete resolution of vulvodynia symptoms with a final VAS score of 0, and the remaining patients had partial improvement. Improvements in daily activities and sexual function were also reported, although xerostomia and drowsiness were common and three patients discontinued treatment due to adverse effects. (Licata et al., 2025)

These findings should not be overgeneralized. Amitriptyline was studied for vulvodynia associated with VLS, not specifically for pruritus, and the evidence consists of a small uncontrolled case series. Nevertheless, the study supports the clinical principle that persistent sensory symptoms in VLS may require a neuropathic pain framework in selected patients.

6. Therapeutic Implications

6.1. Topical corticosteroids remain the foundation of itch control

Potent and ultrapotent topical corticosteroids remain the gold standard for VLS treatment. They reduce inflammation, relieve pruritus in many patients, prevent progression of scarring, and may reduce malignant transformation risk when used appropriately. Guidelines emphasize early diagnosis, adequate induction therapy, and long-term maintenance because genital LS is often lifelong. (De Luca et al., 2023; Kirtschig et al., 2024)

In a pruritus-centered approach, topical corticosteroids should not be regarded merely as anti-inflammatory drugs but as first-line antipruritic therapy for active VLS. Persistent itch during induction therapy should prompt assessment of correct application, sufficient quantity, treatment adherence, irritant avoidance, and exclusion of infection or contact dermatitis. Inadequate response should not automatically lead to repeated unsupervised escalation without diagnostic reassessment.

6.2. Adherence, steroid hesitancy and patient education

Because VLS requires long-term treatment, adherence is central to symptom control. Steroid hesitancy may contribute to undertreatment, recurrent inflammation, persistent pruritus, and progression of scarring. In a large survey of patients with VLS, the mean global steroid-phobia score was 43.02%, and approximately 40% of participants endorsed waiting as long as possible before using topical corticosteroids and stopping as soon as possible. Physician and pharmacist reassurance were the most influential sources for improving comfort with topical corticosteroid use. (Delpero et al., 2023)

However, the relationship between steroid fear and adherence is not uniform. In a prospective study of 50 women with newly diagnosed histologically confirmed VLS treated with a 12-week corticosteroid regimen, the median global TOPICOP score was relatively low at 19.4%, 82.2% of patients adhered to treatment, and neither adherence nor treatment effectiveness was significantly associated with the level of corticosteroid phobia. (Borghi et al., 2025)

Taken together, these studies suggest that steroid hesitancy is clinically relevant but context-dependent. Educational interventions should be practical and individualized: patients should be shown exactly where and how much medication to apply, reassured about appropriate long-term safety, and informed that maintenance therapy is intended to prevent relapse rather than signal treatment failure.

6.3. Emollients, irritant avoidance and vulvar skin care

Barrier-directed care is an important adjunct to anti-inflammatory treatment. Patients should avoid irritants such as fragranced products, harsh soaps, wipes, over-cleansing, tight clothing, and unnecessary topical antimicrobials. Emollients may reduce friction, urine exposure, and mechanical irritation. In patients with urinary incontinence, barrier protection and urogynecological management may be particularly relevant because persistent urine exposure can aggravate burning and itch.

Although emollients do not replace corticosteroids, they may reduce symptom triggers and support barrier recovery. Their role is especially important for patients with persistent irritation despite controlled inflammation.

6.4. Topical calcineurin inhibitors and other anti-inflammatory options

Topical calcineurin inhibitors may be considered second-line or adjunctive therapy when topical corticosteroids are contraindicated, poorly tolerated, or insufficient, but they are not generally superior to ultrapotent corticosteroids as first-line treatment. Their role in itch control is therefore primarily as a corticosteroid-sparing anti-inflammatory option in selected patients. (De Luca et al., 2023; Kirtschig et al., 2024)

Emerging anti-inflammatory strategies, including JAK inhibitors, biologics, and other targeted therapies, remain investigational. Their potential relevance is supported by the cytokine-driven nature of LS, but robust VLS-specific trials are lacking. (Paganelli et al., 2025; Shen et al., 2025)

6.5. Neuromodulatory treatment for persistent pain or vulvodynia-like symptoms

When burning, pain, allodynia, or vulvodynia persists despite adequate disease control, treatment should shift from purely anti-inflammatory escalation to a broader vulvar pain strategy. This may include pelvic floor assessment, sexual counseling, vulvodynia-informed care, and selected neuromodulatory medication.

Amitriptyline has preliminary supportive evidence from a small case series in VLS-associated vulvodynia. The observed improvement in pain and quality-of-life measures suggests that descending pain modulation and peripheral nociceptor modulation may be useful in selected patients. However, adverse effects were common, and controlled studies are required before formal recommendations can be made. (Licata et al., 2025)

Other neuromodulators used in chronic pruritus or vulvodynia, such as gabapentinoids or serotonin–norepinephrine reuptake inhibitors, may be considered only on an individualized basis and should not be presented as evidence-based VLS-specific antipruritic therapy in the absence of direct data.

6.6. Biologic therapy and type 2 inflammation: dupilumab as a signal, not a standard

A case report described a 61-year-old woman with severe vulvar pruritus associated with lichen sclerosus et atrophicus who improved during four months of dupilumab therapy, with pruritus relief and no reported adverse reactions. The authors proposed that IL-4/IL-13 blockade may be relevant to refractory itch in selected non-atopic dermatitis conditions. (Du et al., 2024)

This observation is clinically interesting but should be interpreted cautiously. It does not establish dupilumab as standard therapy for VLS, and current guideline-level evidence for biologics in LS remains limited to isolated reports. Nevertheless, such cases support the hypothesis that refractory itch in some patients may involve cytokine pathways beyond classic Th1 inflammation.

6.7. Laser, photodynamic therapy and regenerative approaches

Energy-based and regenerative therapies, including fractional CO2 laser, photodynamic therapy, platelet-rich plasma, nanofat, stromal vascular fraction, and hyaluronic acid-based approaches, have been studied in VLS with variable quality of evidence. Some reports suggest improvement in symptoms or quality of life, but guidelines remain cautious, particularly regarding laser therapy as a disease-modifying treatment. For a pruritus-focused review, these modalities should be framed as investigational or adjunctive options for selected refractory patients rather than replacements for topical corticosteroids. (Beutler et al., 2025; Gil-Villalba et al., 2023; Kirtschig et al., 2024)

7. Future Directions

Future research should address five priorities.

First, studies should define the phenotype of VLS-related pruritus. This includes intensity, duration, nocturnal worsening, scratch behavior, burning, pain overlap, sexual triggers, urinary exposure, and correlation with clinical activity.

Second, tissue studies should integrate neuroimmune markers, including nerve-fiber density, CGRP, substance P, TRPV channels, mast cells, macrophage subsets, T-cell subsets, keratinocyte-derived cytokines, and perineural inflammation. These should be correlated with itch severity rather than only with histological diagnosis.

Third, trials should include itch as a prespecified endpoint. VAS/NRS itch, sleep disturbance, DLQI/VQLI, sexual function, and patient-priority symptoms should be measured longitudinally.

Fourth, future therapeutic studies should distinguish between active inflammatory itch, irritant/contact-associated itch, and neuropathic/vulvodynia-like symptoms. These phenotypes may require different treatments.

Fifth, targeted therapies should be evaluated rationally. IL-4/IL-13 blockade, JAK inhibition, neuromodulatory agents, and other antipruritic pathways may be relevant, but they require controlled studies before they can be incorporated into standard VLS management.

8. Conclusions

Pruritus is a core symptom of VLS and a major determinant of quality of life, sexual function, psychological burden, and treatment-seeking behavior. The mechanisms of VLS-related itch are likely multifactorial, involving epithelial barrier disruption, chronic inflammation, keratinocyte–immune signaling, altered innervation, neurogenic inflammation, perineural inflammation, and possible neuropathic sensitization in selected patients.

Potent and ultrapotent topical corticosteroids remain the foundation of treatment and are the most evidence-based approach to inflammatory itch control. However, persistent pruritus or burning despite adequate therapy should prompt structured reassessment rather than automatic escalation. Clinicians should

evaluate adherence, application technique, contact or irritant dermatitis, infection, urinary incontinence, genitourinary syndrome of menopause, scarring, vulvodynia, and psychological burden.

Emerging evidence on perineural inflammation, altered innervation, cytokine networks, neuromodulatory therapy, and biologic treatment suggests that refractory symptoms may represent distinct neuroimmune phenotypes. At present, these approaches remain hypothesis-generating. A future precision-medicine approach to VLS-related pruritus will require standardized symptom measurement, tissue-level neuroimmune profiling, and controlled trials focused specifically on itch and sensory outcomes.

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